Myocardial Infarction and Heart Failure

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THE two common cardiac conditions, acute myocardial infarction and cardiac failure, occur frequently in the same patient simultaneously. Their coexistence is not a coincidence but usually there is a causal relationship between them in that one of them precipitates the other one.

It is obvious that acute myocardial infarction, if large enough, may easily precipitate cardiac insufficiency. On the other hand myocardial infarction may develop in the course of heart failure which is the result of other causes. The purpose of this communication is to discuss briefly various aspects of this relationship as found in a series of cases in which autopsies were done.

Patients with congestive cardiac failure caused by hypertension, valvular disease, or other less common causes who have never shown clinical signs of coronary insufficiency, occasionally develop acute myocardial infarction. This may be brought on in different ways. First, myocardial infarction may occur as a result of coronary embolism, the most common source of the embolus being mural thrombosis, which is often present in dilated heart chambers, particularly in the presence of auricular fibrillation. We have recently seen an infarction of the myocardium in an eight year old child with heart failure due to hypertensive heart disease associated with pyelonephritis, where a true coronary embolism came from a mural thrombus. These cases are not common but are important enough to be considered in the differential diagnosis. Another way in which heart failure may precipitate the formation of a myocardial infarct is the production of thrombosis at the site of a previously existing stenosis of one or more branches of coronary arteries. Various factors may contribute to the formation of a thrombus at the site of the stricture: increased tendency to intravascular clotting which occurs in cardiac failure and is possibly enhanced by various drugs, or slowing of the circulation and a fall in blood pressure. In certain circumstances interference with the coronary blood flow may bring about a myocardial infarction even in the absence of a complete occlusion of a major branch of the coronary artery.

Myocardial infarction complicating chronic cardiac failure in hypertensive, rheumatic, luetic and other types of heart disease is a very serious complication. The superimposition of acute damage to the failing myocardium is often fatal within a short time.

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Demonstration at autopsy of a recent infarction of the myocardium in patients dying from chronic cardiac failure due to causes other than coronary heart disease is quite common although the infarction is often unsuspected during life. For some unknown reason myocardial infarction occurring in the course of heart failure develops frequently without pain or any acute equivalent of it. In a series of 130 consecutive cases of recent myocardial infarction taken from our autopsy file, 22 infarcts were found in patients who were in chronic heart failure before the development of the infarction. The clinical symptoms and signs suggested the presence of infarction in only six of these patients, while in the remaining 16 patients it appears that the diagnosis could not have been made.

Thus in the course of chronic heart failure myocardial infarction is a fairly common complication which, along with pulmonary infarction and embolism, ought to be suspected and investigated electrocardiographically if an unexplained change in the condition of the patient takes place.

Myocardial infarction as a complication of chronic heart failure is a very serious and often terminal event. It occurs most often in patients for whom the prognosis is very poor to begin with. On the other hand myocardial infarction occurring in patients whose physical activities showed no significant limitation from cardiac symptoms prior to the development of the infarction is referred to as "primary" myocardial infarction. Heart failure developing secondarily in the course of myocardial infarction belongs to an entirely different class of cardiovascular events. Myocardial infarction often runs the course of an acute illness with complete recovery, and the presence or absence of heart failure and of various other complications in its course is of immediate and great practical importance.

Physiologically speaking, heart failure is a direct consequence of acute myocardial infarction and is present in every case of sizable infarction of the myocardium. Destruction of a portion of the myocardium always affects the contraction of the heart and causes a fall in cardiac output, dilatation of the affected chamber (almost always the left ventricle), and increased pressure in the left auricle and pulmonary veins, even if these events take place only momentarily. Dilatation of the left ventricle acts as a stimulus for a more forceful contraction of the non-infarcted muscle fibers, and the effective output may be reestablished if favorable conditions exist.

From the clinical standpoint it is important to know what role this decompensation plays in the course of myocardial infarction. Patients who have had no significant cardiac symptoms prior to the development of the myocardial infarction can be

divided into several groups, the classification depending upon the presence and degree of cardiac failure resulting from the infarct. First, there are patients in whom no clinical signs of left ventricular failure are evident, although in almost every case latent left ventricular failure can be demonstrated by the use of special methods.[3] Or, patients may show evidence of mild left sided failure, such as basilar rales, gallop rhythm and mild to moderate dyspnea, which are often transitory. Still other patients may have severe left ventricular failure: pulmonary edema, marked dyspnea, orthopnea and cyanosis. Secondarily to left ventricular failure the action of the right ventricle may become insufficient and increased venous pressure and peripheral edema may appear. Finally, in some patients the predominant clinical picture is that of shock.

What is the effect of these various forms of circulatory failure upon the course and mortality from acute myocardial infarction? In order to attempt to answer this question a study was made of a series of 95 cases of fatal myocardial infarction in which autopsies were done. These cases were unselected, but in all of them myocardial infarction was the primary disease and all the patients died within six weeks from the onset of the infarction.

The incidence of the three forms of circulatory failure was as follows: left ventricular failure was present in 65 cases; shock occurred in 20, and right ventricular failure in 17 cases.

Left ventricular failure, by far the commonest clinical manifestation of circulatory derangement due to myocardial infarction, appears, however, to be of least prognostic significance. Of the 65 patients in whom it was present, only 14 died of progressive cardiac failure, while in the remaining 51 the signs of left ventricular failure cleared up or remained mild, and the patients died of secondary complications unrelated to cardiac failure.

Right ventricular failure occurred in 17 patients. In contrast to left ventricular failure, systemic congestive failure occurred late for the most part and was invariably fatal. Only one patient developed right ventricular failure within the first week of the illness, and that was in a case of myocardial infarction complicated by perforation of the interventricular septum. This condition is known to be associated with marked right ventricular failure because the increased volume of blood in the failing left ventricle is shunted through the septal defect directly into the right ventricle, thus eliminating pulmonary congestion and leading to systemic congestion. In the remaining 16 patients who had right ventricular failure, it developed secondarily to left sided failure, usually in the second or third week. It was noticeable that in half of them postmortem examination revealed, in addition to the myocardial infarction, the presence of multiple pulmonary infarcts, and in these cases right sided failure was most severe. These findings suggest a causal relationship between pulmonary emboli and infarcts and right ventricular failure in the course of myocardial infarction. Evidently right ventricular strain due to pulmonary emboli, added to pulmonary hypertension caused by left ventricular failure, may bring about failure of the right ventricle although either of these two factors alone might be insufficient for the development of clinical manifestations of systemic venous congestion. It is believed that knowledge of this relationship may be of some diagnostic value through directing attention to the presence of pulmonary emboli in patients who show marked right ventricular failure. This may be of only academic interest, however, because the prognosis is very unfavorable if right ventricular failure develops in the course of myocardial infarction.

Shock occurred in 20 patients. It was an early complication, occurring with or shortly after the initial attack of pain. In 19 patients the course was progressively downhill. In one patient the signs of shock cleared up but the patient died later from an unrelated complication.

The seriousness of the condition of the patients who develop shock in the course of myocardial infarction need not be emphasized: they present perhaps the most dramatic clinical pattern of this condition. The cause of shock is not known. It represents "forward" failure of the circulation, namely, a sudden marked decrease in cardiac output with clinical manifestations of intense vasoconstriction, a compensatory attempt to maintain reasonable blood pressure as long as possible in spite of the falling cardiac output. The mechanism of traumatic and surgical shock is known to be peripheral and not cardiac in origin, namely, failure of sufficient quantity of blood to reach the heart. According to Starling's law of the heart, the diminished venous return causes a fall in cardiac output, so that the heart is involved in this process passively without true insufficiency of the myocardium. It has been suggested[1] that the shock of myocardial infarction is cardiogenic: the damaged left ventricle fails to maintain the output, and the suddenness of this may produce the clinical picture of forward failure more prominently than that of congestive phenomena. This theory has many attractive points, but it is based on speculation and is thus far not proven by any studies of circulation. On the other hand, there are some points favoring the theory of peripheral origin of shock in myocardial infarction, possibly a reflex pooling of the blood in the periphery with a secondary fall of the venous return and cardiac output. This alternative view, if confirmed, implies that shock is a complication and not a direct consequence of myocardial insufficiency, and emphasizes the importance of active therapeutic measures.

It is very instructive to present the immediate causes of death in the series of 95 patients with myocardial infarction. As has been pointed out, these were patients with "primary" myocardial infarction, and therefore were well suited for the consideration of the immediate effects of the acute infarct upon the circulation. Theoretically, myocardial infarction can bring about death in two ways: it can damage the myocardium to such an extent that the effective maintenance of the circulation be-

comes impossible; or it may lead to secondary complications which prove fatal. In the first group one would expect either immediate death or the development of cardiac insufficiency with continuous progression until death. In the second group cardiac failure is often absent, and this favorable course suggests that the patients might have recovered if the complications could have been avoided.

In this series the immediate causes of death, or the most important factor immediately responsible for death, were as follows: progressive cardiac insufficiency, 14 cases; shock, 14 cases; sudden death (ventricular fibrillation?), 24 cases; thromboembolic phenomena, 15 cases; cardiac rupture, eight cases; secondary coronary occlusion, five cases; incidental complication, 15 cases.

These figures indicate that cardiac insufficiency is not the most important cause of death in myocardial infarction. All the patients in this series had been hospitalized and had lived for at least several hours after the onset of the illness. No cases in which the patients died within the first hour or two are included in the statistics, which are for the most part based on hospital material. It is quite probable that patients who die before having had time to reach the hospital represent the most serious cases from the standpoint of circulatory derangement. It has been estimated that the immediate mortality from myocardial infarction of the "primary" type is below 20 per cent. In thoses cases in which death occurs, it is doubtful that it occurs in more than one third within the first few hours. Thus one can visualize the effective compensatory measures occurring early in myocardial infarction. In 90 per cent or more of patients with myocardial infarction the circulation is effectively reestablished, and these patients are candidates for recovery from the immediate attack. Many of them can recover completely for indefinite periods of time. Some of them, however, succumb to various complications developing within the first few weeks following the attack. The small number of patients who die of circulatory failure either develop fatal decompensation at the onset or proceed into progressive cardiac failure.

It is important to realize that more patients die in the course of acute myocardial infarction from secondary complications than from irreparable cardiac damage. Theoretically the presence of heart failure and a progessively downhill course indicate irreparable cardiac damage. Patients in whom these conditions exist represent the irreducible mortality from myocardial infarction. Patients who die even though an effective circulation can be reestablished despite the damage to the heart, should be considered as dying prematurely and unnecessarily. They die because present-day methods of prevention or treatment of various complications are inadequate. The three types of complications responsible for most premature deaths are thromboembolic phenomena, shock, and fatal arrhythmias. The nature of each of these complications is such that prevention or effective treatment appears within the realm of possibility. So far, the only hopeful approach toward the solution of this problem is the recent introduction of anticoagulants in the treatment of myocardial infarction. Not yet proven completely safe and effective, and requiring elaborate technical facilities, this method may eventually become routine treatment of myocardial infarction.

The need for finding better methods of treatment of myocardial infarction is very urgent and its importance is emphasized by two facts. First, the number of persons at stake is very large. It has been estimated[2] recently that more than 100,000 persons die annually from coronary occlusion in the United States. The second point is the ever-increasing realization in recent years that persons who recover from acute myocardial infarction need no longer be considered chronic cardiac invalids. A large proportion of them regain normal cardiac function and enjoy indefinite periods of healthy and useful life after recovery from myocardial infarction.

The large number of potentially preventable deaths from acute myocardial infarction among patients included in the present series indicates the possibility that the immediate mortality from myocardial infarction may be reduced considerably in the future.

SUMMARY

The relationship between myocardial infarction and cardiac failure manifests itself in two ways: myocardial infarction may appear as a complication of chronic cardiac failure, or heart failure may appear as a sequence of acute myocardial infarction in patients who had no disturbance of cardiac function prior to the formation of the infarct.

Various forms of circulatory failure appearing in the course of myocardial infarction are discussed. Left ventricular failure is the most common, but most often is a self-limited, reversible process. Right ventricular failure is relatively uncommon, is often associated with pulmonary complications, and is almost invariably fatal. Shock, also a serious complication of myocardial infarction, is usually fatal in these circumstances.

An analysis of a series of cases of fatal myocardial infarction revealed that severe progressive cardiac failure was the primary cause of death in less than 25 per cent of the patients in the series. Other patients died as a result of secondary complications developing in the first few weeks after the coronary occlusion.

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